

Transient myopia after aspirin

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Transient myopia occurs after a variety of noxious stimuli to the eye. Although individual case reports are quite common, the condition is apparently only rarely seen. It has been noted as a toxic reaction to various drugs, in particular sulphonamides (especially the earlier and now obsolete preparations) and the chemically related oral diuretics. Mattsson (1952) collected over fifty references to transient myopia due to sulphonamides and Muirhead and Scheie (1960) reviewed eight cases due to acetazolamide. In recent years the condition has also been reported after ethoxolamide (Beasley, 1962), hydrochlorothiazide (Beasley, 1961), Chlorthalidone (Michaelson, 1962), and prochlorperazine (Yasuna, 1962), and there have been further reports after sulphonamides (Maddelena, 1968) and acetazolamide (Galín, Baras, and Zweifach, 1962).

Transient myopia is a well recognized phenomenon in diabetes and after blunt injuries to the eye (Duke-Elder, and Abrams, 1970); it has been observed after light coagulation (Boulton, 1973), and may even occur during menstruation (Jampolsky and Flom, 1953). It is uncertain how the myopia is caused, but other significant features frequently recorded are transient retinal oedema, anterior displacement of the lens with anterior chamber shallowing, and transient closed-angle glaucoma.

A case is described below in which transient myopia, unaffected by cycloplegia, occurred after the administration of aspirin (acetylsalicylic acid). There was associated shallowing of the anterior chamber, an increase in the antero-posterior diameter of the lens, a slight rise in the intraocular pressure, and some retinal oedema. A search of the literature revealed only one previous report of transient myopia after aspirin (Korol, 1962), which was associated with a transient rise in intraocular pressure; no definite conclusions were reached concerning the mechanism.

Case report

A 39-year-old man with previously normal vision presented at the Bristol Eye Hospital complaining of blurred vision since waking that day. The previous evening, hoping to ward off a cold, he had taken eight aspirin tablets with a small glass of whisky (2.7 g. acetylsalicylic acid in all).

Examination

The visual acuity was 6/60 in the right eye and 6/36 in the left, but improved on refraction to 6/6 in each eye with -3.25 D sph. The anterior chambers were shallow, the pupil reactions normal, and the ocular media clear. The intraocular pressures were 28 mm.Hg in the right eye and 29 mm.Hg in the left by applanation. Gonioscopy showed very narrow angles with possible irido-corneal contact superiorly.

A general physical examination showed nothing abnormal and there were no signs of a cold or other virus infection. It was felt that ciliary spasm and forward displacement of the iris-lens diaphragm was the most likely explanation for the myopia and therefore, in spite of the narrow anterior chamber angle, cycloplegic drops (cyclopentolate) were instilled and the patient carefully observed. One hour later the retinoscopy remained unchanged and the myopia persisted. The anterior chamber remained shallow but the intraocular pressures had fallen to 24 mm.Hg in each eye. The anterior

chamber depths were measured with the depth gauge attachment to the Haag-Streit slit lamp and were found to be 2.2 mm. in both eyes. The same instrument was used to measure the antero-posterior lens diameter. (Owing to magnification effects this would not be a true reading, but would show changes in the lens diameter on serial recordings.) The apparent diameter was 5.2 mm. in both eyes. Fundus examination after mydriasis showed slight but generalized retinal oedema, with pallor of the retina and apparent increase in the retinal thickness on slit-lamp funduscopy.

No treatment was given and 2 days later the visual acuity was 6/5 unaided in the right eye and 6/18 unaided in the left (improving to 6/5 with -0.5 D sph., -0.5 D cyl., axis 60°). The anterior chambers appeared to be normal and the intraocular pressures were 12 mm.Hg in each eye. Cycloplegic drops were again instilled and measurements taken of the anterior chamber depth and antero-posterior lens diameter. In the right eye the anterior chamber depth was 3.0 mm., apparent lens diameter 4.85 mm., and in the left eye 2.7 mm. and 5.0 mm. respectively. The fundi were normal. A week later the visual acuity in both eyes was 6/5 unaided and the anterior chamber depth and antero-posterior lens diameter in the left eye were similar to those seen earlier in the right.

Discussion

Transient myopia after drugs is thought to be a toxic phenomenon although, in certain instances, the dose involved is so low that presumably there must be a hypersensitivity or allergy to the causative agent (Maddelena, 1968).

It may often be difficult to implicate any one agent—for instance, in this case it could be argued that the myopia was caused by the whisky or the aborted “cold” and not by the rather large dose of aspirin; in a previous report of transient myopia ascribed to menstruation (Jampolsky and Flom, 1953), the patient may inadvertently have taken an oral preparation containing diuretics for premenstrual tension.

In spite of numerous reports about toxic myopia, little is known for certain of its mechanism. The various factors which could make an eye temporarily myopic have already been listed (Jampolsky and Flom, 1953). In a certain proportion of cases the myopia is abolished by cycloplegia, thus suggesting that the cause is ciliary spasm, but in most cases as in that reported above cycloplegia does not affect the myopia.

The myopia has usually been ascribed to changes in the position or refraction of the lens and in this case there was objective evidence of both these changes. Anterior chamber shallowing, sometimes to the extent of causing closed-angle glaucoma, and retinal oedema, have both been frequently noted in association with transient toxic myopia.

One hypothesis which could explain all these findings is that the permeability of the retinal vessels is temporarily increased by the toxic agent, thus causing retinal oedema and an escape of osmotically active substances into the vitreous and a consequent increase in vitreous volume. This would cause a forward displacement of the lens. If the osmotic changes in the vitreous were transmitted to the lens, this also would increase in size (the transient myopia of diabetes is usually ascribed to an osmotic process in the lens).

An alternative theory proposed by Mattsson (1950) is that the oedema is primarily located in the ciliary body, thus causing a relaxation of the zonule so that the lens assumes its accommodated form and is also displaced forwards. This, however, would not account for the degree of forward shift of the lens that may occur (Jampolsky and Flom, for instance, considered the anterior chamber in their case to be only 0.5 mm. deep), and it is difficult to see how an amplitude of accommodation is preserved when the lens is in its accommodated form and why there should be no reversal of the myopia from cycloplegics.

It is interesting, also, to consider the transient myopia which may follow extensive light coagulation (Boulton, 1973). In this condition there is a marked increase in vitreous volume, demonstrated by changes in the position of the vitreous face in aphakic eyes, with forward

displacement of the iris-lens diaphragm in phakic eyes. The speed of progression and regression of the myopia is similar to that reported here and to that usually noted for drug-induced myopia. However, the degree of myopia is less and little or no significant change is noted within the lens. This may partly be due to the greater age of most patients having extensive light coagulation and, hence, more rigid lenses.

Similarly, transient anterior chamber shallowing has been noted after retinal vein occlusion (Hyams and Neumann, 1972; Grant, 1973) and, although the refraction was not measured, one would expect some degree of myopia after a forward lens displacement. Extensive light coagulation of the retina and acute retinal vein occlusion would both produce considerable retinal oedema and it may be that the condition of forward displacement of the lens with transient myopia is a common reaction to various stimuli causing retinal oedema. It is reasonable to assume that the presumed osmotic changes in the vitreous may also affect the lens in younger patients. This could be one possible explanation of the transient myopia after trauma, which is often associated with retinal oedema, although trauma is such a non-specific insult to the eye that other mechanisms may also be involved.

The coincident glaucoma may present some difficulty in treatment. The case described here improved with mydriatics, although they were originally administered in the false assumption that ciliary spasm was present, presumably because the element of pupil block was greater than that of angle block. This point and the need to avoid surgery has already been discussed by Grant (1973).

Another interesting corollary is that, as transient anterior chamber shallowing can be caused by stimuli of such varying nature, it is possible that this mechanism may have some significance in the pathogenesis of acute closed-angle glaucoma. Hitherto, interest has mainly been centred on factors causing transient pupil changes rather than factors causing transient anterior chamber depth changes.

Summary

A case of transient myopia after ingestion of aspirin is described. Anterior chamber shallowing, changes in the lens, a rise in the intraocular pressure, and retinal oedema were also observed. It is suggested that drug-induced transient myopia is caused by a change in the osmotic pressure in the vitreous, and some comparison is made with other causes of transient myopia.

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